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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/494,096	01/28/2000	Gary A. Bannon	HS 102	3034

7590 03/12/2002

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EXAMINER	
HUYNH, PHUONG N	
ART UNIT	PAPER NUMBER
1644	12

DATE MAILED: 03/12/2002

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/494,096	BANNON ET AL.	
	Examiner	Art Unit	
	" Neon" Phuong Huynh	1644	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *Three* MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *31 July 2001* .

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-36* is/are pending in the application.

4a) Of the above claim(s) *1-29 and 33-36* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *30-32* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☒ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s) *3 & 8* .
4) ☐ Interview Summary (PTO-413) Paper No(s). _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: .

DETAILED ACTION

1. Claims 1-36 are pending.

2. Applicant's election without traverse of Group XVI, claims 30-32 drawn to a nucleic acid encoding modified allergen, filed 7/23/01, is acknowledged.

3. Claims 1-29 and 33-36 are withdrawn from further consideration by the examiner, 37 C.F.R. 1.142(b) as being drawn to non-elected inventions.

4. Claims 30-32 are being acted upon.

5. The drawings, filed 1/28/00, are not approved. Please see enclosed PTO 948, Notice of Draftsperson's Patent Drawing Review. Appropriate action is required.

6. The references crossed out on the form PTO 1449, filed 5/15/00, have not been considered because applicant failed to supply said references.

7. The disclosure is objected to because of the following informalities: (1) typographical error on page 14 second full paragraph, "IL 12, IL 16, IL 18, Ifn-ξ" should have been "IL-12, IL-16, IL-18 and IFNγ"; (2) "igE" on page 27, line 13 should have been "IgE"; (3) The paper copy of the sequence listing and the computer readable form do not match with the sequence description in the specification. The specification on page 15 line 8 from the bottom of the page discloses that the nucleotide sequences of Ara h1, Ara h2 and Ara h3 are SEQ ID NOS: 1, 3 and 5, respectively, while the paper copy of the sequence listing and the computer form disclose that the nucleotide sequences of Ara h1, Ara h2 and Ara h3 are SEQ ID NOS: 2, 4 and 6, respectively; (4) the specification on pages 1-21 do not have line number on the left margin while pages 23-32 have line number on the left margin. Appropriate correction is required.

8. The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

9. Claims 30-32 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling only for (1) three isolated nucleotide molecules consisting of SEQ ID NO: 1, 3 and 5 from peanut allergens Ara h 1, Ara h 2 and Ara h3, respectively, vector and host cell for making recombinant peanut allergen Ara h 1, Ara h 2 and Ara h3 polypeptides consisting of SEQ ID NO: 2, 4 and 6, respectively (See page 18) and (2) amino acid substitution of said polypeptide produces modified Ara h1, Ara h2 and Ara h3 peptides such as the ones listed on page 23, lines 15-18, Table 4, 5, 6 and page 28, line 14-17 wherein the modified Ara h1, Ara h2 and Ara h3 peptides bind less IgE than unmodified recombinant allergen mentioned above for immunotherapy, does not reasonably provide enablement for (1) *any* nucleotide molecule encoding *any* modified allergen which is less reactive with IgE comprising at least one IgE binding site present in the allergen modified by at least one amino acid change so that the site no longer binds IgE but wherein the modified allergen activates T cells, (2) *any* nucleotide molecule encoding *any* modified allergen in a vector for expression in *any* recombinant host, (3) *any* nucleotide molecule for causing a site specific mutation in *any* gene encoding any protein which yields *any* modified allergen which is less reactive with IgE comprising at least one IgE binding site present in the allergen modified by at least one amino acid change so that the site no longer binds IgE, but wherein said modified allergen activates T cells. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in **scope** with these claims.

Factors to be considered in determining whether undue experimentation is required to practice the claimed invention are summarized *In re Wands* (858 F2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988)). The factors most relevant to this rejection are the scope of the claim, the amount of direction or guidance provided, the lack of sufficient working examples, the unpredictability in the art and the amount of experimentation required to enable one of skill in the art to practice the claimed invention. The specification disclosure is insufficient to enable one skilled in the art to practice the invention as broadly claimed without an undue amount of experimentation.

The specification discloses only three isolated nucleotide molecules consisting of SEQ ID NOS: 1, 3 and 5 of peanut allergens Ara h 1, Ara h 2 and Ara h3, respectively, vector and host cell for producing recombinant Ara h 1, Ara h 2 and Ara h3 polypeptides consisting of SEQ ID NOS: 2, 4 and 6, respectively (See page 18). The specification discloses that only the specific amino acid substitution within the IgE binding epitope of Ara h1 polypeptide of SEQ ID NO: 2

such as the ones listed in Table 4 would lead to a reduction in IgE binding. Likewise, a specific single amino acid substitution within the IgE binding epitope of Ara h2 polypeptide of SEQ ID NO: 4 such as the ones listed in Table 5 would bind less IgE and stimulate T cell than unmodified recombinant Ara h2. Again, only the specific amino acid substitution such as the ones listed in Table 6 within the IgE epitope of Ara h3 polypeptide of SEQ ID NO: 6 would bind less IgE for recombinant allergen for desensitization immunotherapy.

Other than the specific polynucleotide molecules mentioned above, the specification fails to provide *any* guidance as how to make and use *any* "nucleotide molecule" encoding *any* modified allergen which is less reactive or no longer binds IgE and yet activates T cell for immunotherapy. The specification fails to provide guidance as to which nucleotide(s) within *any* of the undisclosed polynucleotide molecule that would encode the critical IgE binding epitopes of *any* allergens other than Ara h1, Ara h2 and Ara h3. The specification also fails to provide guidance as to which nucleotide(s) within any of the undisclosed polynucleotide molecule that can be change and whether after modification would encode a modified allergen that would decrease IgE binding and increase T cell proliferation for desensitization immunotherapy.

Ferreira *et al* teach nucleotide molecules for site-directed mutagenessis in a gene encoding a protein such as the major hazel pollen allergen Cor a 1/16 which yields a modified allergen Cor a 1/16 T10 that fails to be less reactive with IgE wherein the modified hazel pollen allergen comprises at least one amino acid change such as proline to threonine (See page 128, DNA construct, Table 1, T1 P10 to T, page 132, third paragraph from bottom, in particular). It is known in the art that even a single amino acid changes or differences in a protein's amino acid sequence can have dramatic effects on the protein's function. Fasler *et al.* (PTO 892) teach that peptides derived from house dust mite Der p1 are modified by single amino acid substitutions at positions 173, 175, 176, 180 and 181 with alanine or glycine failed to induce Der p1 specific T cell proliferation and IL-2, IL-4 and IFN-γ production. Fasler *et al.* further teach that substituting a neutral Asn residue at position 173 with a basic Lysine, a hydrophobic Try, Ile, an acidic Asp or a hydrophilic residue serine also did not induce T cell proliferation and cytokine production. However, substitution amino acid positions other than 173, 175, 176, 180 and 181 induces normal or only slightly reduced proliferative responses and cytokine production by T cells (page 524, in particular). Burks *et al.* (PTO 892) teach a modified allergen from peanut Ara h1 where the immunodominant IgE binding epitope of Ara h1 is modified by amino acid substitution at position 1, 3, 4 and 17 with alanine or glycine reduced IgE binding. In contrast, substituting an

alanine for glutamine residue at position 31 leads to an increase IgE binding. Burks *et al.* further teach that "there is no obvious position within each peptide that when mutated, would result in loss of IgE binding and there was no consensus in the type of amino acid that, when changed to alanine, would lead to loss of IgE binding" (See page 338, in particular). Stanley et al (PTO 892) teach a modified peanut allergen Ara h2 by amino acid substitution with alanine at position 67, 68 or 69 significantly reduced IgE binding while substitution of serine residue at position 70 leads to an increased in IgE binding. Stanley et al also teach that in general, "each epitope could be mutated to a non-IgE binding peptide by the substitution of an alanine for a single amino acid residue. However, there was no obvious position within each peptide that, when mutated, would result in loss of IgE binding. Furthermore, there was no consensus in the type of amino acid that, when changed to alanine, would lead to loss of IgE binding" (See page 251, in particular). Skolnick *et al* teach that sequence-based methods for function prediction are inadequate and knowing a protein's structure does not tell one its function (See abstract, in particular). Colman *et al* teach that a single amino acid changes within the interface of antibody-antigen complex can abolish the antibody-antigen interaction or binding entirely (See page 33, in particular). Since there is no obvious position within each polypeptide that, when mutated, would result in loss of IgE binding, it follows that the specific nucleotide within the polynucleotide molecule encoding said polypeptide (modified allergen) would require guidance. Given the indefinite number of undisclosed nucleotide molecule, it is unpredicatable which undisclosed polynucleotide molecule that encodes a modified allergen that is less reactive with IgE, or no longer binds IgE and activates T cells, in turn, would be useful for producing the modified allergen for desensitization immunotherapy. Since the nucleotide molecule as recited in claim 30 is not enabled, it follows that any molecule of claim 30 in a vector for expression in a recombinant host is not enable.

With regard to "a nucleotide molecule for causing a site-specific mutation in a gene encoding a protein which yields a modified allergen", there is no guidance and working examples in the specification as filed that just any nucleotide molecule can cause a site-specific mutation in *any* gene encoding *any* protein which yields *any* modified allergen. Furthermore, there is no guidance and working examples that even one nucleotide molecule can cause a site-specific mutation in a gene encoding peanut allergen such as Ara h 1, Ara h2 and Ara h3. Given that the gene encoding the allergen protein is not defined and there is no guidance as to which nucleotide position within said gene encoding an amino acid sequence that comprises at least one IgE binding site, the success of predicting which nucleotide molecule would bind to said gene for

causing a site-specific mutation that would yield a modified allergen having at least one amino acid substitution so that the modified allergen no longer binds IgE but activates T cells is null.

For these reasons, the specification as filed fails to enable one skill in the art to practice the invention as broadly as claimed without undue amount of experimentation. In re wands, 858 F.2d at 737, 8 USPQ2d at 1404 (Fed. Cir. 1988), the decision of the court indicates that the more unpredictable the area is, the more specific enablement is necessary. As such, further research would be required. In view of the quantity of experimentation necessary, the insufficient number of working examples, the unpredictability of the art, the insufficient guidance and the breadth of the claims, it would take undue trials and errors to practice the claimed invention.

10. Claims 30-32 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor, at the time the application was filed, had possession of the claimed invention.

The specification does not reasonably provide a **written description** of (1) *any* nucleotide molecule encoding *any* modified allergen which is less reactive with IgE comprising at least one IgE binding site present in the allergen modified by at least one amino acid change so that the site no longer binds IgE but wherein the modified allergen activates T cells, (2) *any* nucleotide molecule encoding *any* modified allergen in a vector for expression in *any* recombinant host, (3) *any* nucleotide molecule for causing a site specific mutation in *any* gene encoding any protein which yields a modified allergen which is less reactive with IgE comprising at least one IgE binding site present in the allergen modified by at least one amino acid change so that the site no longer binds IgE, but wherein said modified allergen activates T cells.

The specification discloses only three isolated nucleotide molecules consisting of SEQ ID NOS: 1, 3 and 5 of peanut allergens Ara h 1, Ara h 2 and Ara h3, respectively, vector and host cell for producing recombinant Ara h 1, Ara h 2 and Ara h3 polypeptides consisting of SEQ ID NOS: 2, 4 and 6, respectively (See page 18). The specification discloses that only the specific amino acid substitution within the IgE binding epitope of Ara h1 polypeptide of SEQ ID NO: 2 such as the ones listed in Table 4 would lead to a reduction in IgE binding. Likewise, a specific single amino acid substitution within the IgE binding epitope of Ara h2 polypeptide of SEQ ID NO: 4 such as the ones listed in Table 5 would bind less IgE and stimulate T cell than unmodified recombinant Ara h2. Again, only the specific amino acid substitution such as the ones listed in

Table 6 within the IgE epitope of Ara h3 polypeptide of SEQ ID NO: 6 would bind less IgE for recombinant allergen for desensitization immunotherapy.

With the exception of the specific polynucleotide molecules mentioned above, there is insufficient written description about the structure associated with function of (1) *any* nucleotide molecule encoding *any* modified allergen, (2) *any* nucleotide molecule encoding *any* modified allergen in a vector for expression in a recombinant host, and (3) *any* nucleotide molecule for causing a site specific mutation in a gene encoding a protein which yields a modified allergen which is less reactive with IgE comprising at least one IgE binding site present in the allergen modified by at least one amino acid change so that the site no longer binds IgE, but wherein the modified allergen activates T cells.

Given the lack of a written description of *any* additional representative species of polynucleotide molecule encoding a modified allergen and *any* nucleotide molecule for causing a site specific mutation in a gene encoding a protein which yields a modified allergen, one of skill in the art would reasonably conclude that the disclosure fails to provide a representative number of species to describe the genus. Thus, Applicant was not in possession of the claimed genus. *See University of California v. Eli Lilly and Co. 43 USPQ2d 1398.*

Applicant is directed to the Revised Interim Guidelines for the Examination of Patent Applications Under the 35 U.S.C. 112, ¶ 1 "Written Description" Requirement, Federal Register, Vol. 66, No. 4, pages 1099-1111, Friday January 5, 2001.

11. The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter, which the applicant regards as his invention.

12. Claims 31 and 32 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

The recitation of "a nucleotide molecule for causing a site specific mutation in a gene encoding a protein which yields a modified allergen" in claim 32 is ambiguous and indefinite. The specification does not define "a nucleotide molecule for causing a site specific mutation in a gene encoding a protein which yields a modified allergen". One of ordinary skill in the art cannot appraise the metes and bounds of the claimed invention.

The recitation of "a recombinant host" in claim 31 is ambiguous and indefinite. The specification on page 11, lines 15-17 discloses **host cell** such as bacteria, yeast, and baculovirus-insect cell systems for production of recombinant or modified allergens.

13. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office Action:

A person shall be entitled to a patent unless –
(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

(e) the invention was described in a patent granted on an application for patent by another filed in the United States before the invention thereof by the applicant for patent, or on an international application by another who has fulfilled the requirements of paragraphs (1), (2), and (4) of section 371(c) of this title before the invention thereof by the applicant for patent.

14. The changes made to 35 U.S.C. 102(e) by the American Inventors Protection Act of 1999 (AIPA) do not apply to the examination of this application as the application being examined was not (1) filed on or after November 29, 2000, or (2) voluntarily published under 35 U.S.C. 122(b). Therefore, this application is examined under 35 U.S.C. 102(e) prior to the amendment by the AIPA (pre-AIPA 35 U.S.C. 102(e)).

15. Claims 30-32 are rejected under 35 U.S.C. 102(b) as being anticipated by Wiedemann *et al* (J Biol Chem 271(47): 29915-29921; Nov 1996, PTO 892).

Wiedemann *et al* teach nucleotide molecules such as the DNA inserts of clones 4, 25 and 35 from the plasmid pMW175 (vector) in host cell *E coli* that encode modified allergens such as modified Birch Profilin (See page 29916, column 1, in particular). The reference modified allergen is less reactive with IgE and has at least one amino acid change such as Phe 44 to Tyr in clone 4, Gln 47 to Glu in clone 25, Gln 47 to Asn in clone 35 that was expressed in host cell such E coli (See page 29916, column 1, Expression and Purification of recombinant Birch Profilin, column 2, Site-directed Mutagenesis of Birch Profilin, in particular). Wiedemann *et al* further teach nucleotide molecule such as primers (See page 29916, column 2, Site-directed Mutagenesis of Birch Profilin, in particular) that cause a site specific mutation in a gene encoding the modified Birch Profilin allergen having at least one amino acid change that no longer binds IgE (See page 29920, column 1, and Figs 7 and 8, in particular). While the reference is silent that the reference nucleotide molecule encoding a protein which yields a modified allergen that activates T cells,

the functional properties would be an inherent property of said nucleotide molecule. Therefore the claimed nucleotide molecule appears to be the same as the prior art nucleotide molecule. Since the Patent Office does not have the facilities for examining and comparing the antibodies of the instant invention to those of the prior art, the burden is on applicant to show that the prior art antibody is different from the claimed nucleotide molecule. See In re Best, 562 F.2d 1252, 195 USPQ 430(CCPA 1977). Thus, the reference teachings anticipate the claimed invention.

16. Claims 30-31 are rejected under 35 U.S.C. 102(e) as being anticipated by US Pat No. 5,840,316 (Nov 1998, PTO 892).

The '316 patent teaches nucleic acid sequences (nucleotide molecule) encoding a modified (derivative) allergen such as ryegrass pollen allergen which stimulates minimal amounts of IgE, binding of IgE and activates T cell response such as T cell proliferation, and/or lymphokine secretion (See column 7, lines 23-65, column 8 lines 66 bridging column 9, lines 1-5, Fig 3b-c, Fig 10a-10b, in particular). The reference modified allergen has at least one amino acid substitution within the one of the IgE binding site (See column 14, lines 61-67, Table 3, in particular). The '316 patent further teaches a vector such as pTRC and host cells such as E coli for producing recombinant protein (See column 11, lines 16-65, in particular).
Thus, the reference teachings anticipate the claimed invention.

17. Claims 30-31 are rejected under 35 U.S.C. 102(b) as being anticipated by Nishiyama *et al* (Molecular Immunology 32(14/15): 1021-1029, Oct 1995; PTO 892).

Nishiyama *et al* teach a nucleotide encoding a modified allergen such as Der f2 in the expression plasmid pGEMEX1 carrying various single amino acid substitution such as from Asn 10 to Ala (See page 1023, Fig 1, column 2, in particular) in host cell such as *E coli* wherein the modified allergen causes a marked decrease in IgE binding (See page 1026, Effect of a single amino acid substitution on IgE binding, in particular). Nishiyama *et al* further teach various nucleotide molecules for causing a site specific mutation in a gene encoding a major mite allergen Der f2 (See page 1022, Tables 1 & 2, column 2, Site-directed mutagenesis, in particular). While the reference is silent that the reference nucleotide molecule encoding a protein which yields a modified allergen that activates T cells, the functional properties would be an inherent property of said nucleotide molecule. Therefore the claimed nucleotide molecule appears to be the same as the prior art nucleotide molecule. Since the Patent Office does not have the facilities for

examining and comparing the antibodies of the instant invention to those of the prior art, the burden is on applicant to show that the prior art antibody is different from the claimed nucleotide molecule. See In re Best, 562 F.2d 1252, 195 USPQ 430(CCPA 1977). Thus, the reference teachings anticipate the claimed invention.

INFORMATION ON HOW TO EFFECT DRAWING CHANGES

18. **Correction of Informalities -- 37 CFR 1.85**

New corrected drawings must be filed with the changes incorporated therein. Identifying indicia, if provided, should include the title of the invention, inventor's name, and application number, or docket number (if any) if an application number has not been assigned to the application. If this information is provided, it must be placed on the front of each sheet and centered within the top margin. If corrected drawings are required in a Notice of Allowability (PTOL-37), the new drawings **MUST** be filed within the **THREE MONTH** shortened statutory period set for reply in the "Notice of Allowability." Extensions of time may NOT be obtained under the provisions of 37 CFR 1.136 for filing the corrected drawings after the mailing of a Notice of Allowability. The drawings should be filed as a separate paper with a transmittal letter addressed to the Official Draftsperson.

Corrections other than Informalities Noted by Draftsperson on form PTO-948.

All changes to the drawings, other than informalities noted by the Draftsperson, **MUST** be made in the same manner as above except that, normally, a highlighted (preferably red ink) sketch of the changes to be incorporated into the new drawings **MUST** be approved by the examiner before the application will be allowed. No changes will be permitted to be made, other than correction of informalities, unless the examiner has approved the proposed changes.

Timing of Corrections

Applicant is required to submit acceptable corrected drawings within the time period set in the Office action. See 37 CFR 1.85(a). Failure to take corrective action within the set period will result in **ABANDONMENT** of the application.

19. No claim is allowed.

20. Any inquiry concerning this communication or earlier communications from the examiner should be directed to "Neon" Phuong Huynh whose telephone number is (703) 308-4844. The examiner can normally be reached Monday through Friday from 9:00 am to 6:00 p.m. A message may be left on the examiner's voice mail service. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Christina Chan can be reached on (703) 308-3973. Any inquiry of a general nature or relating to the status of this application should be directed to the Technology Center 1600 receptionist whose telephone number is (703) 308-0196.

21. Papers related to this application may be submitted to Technology Center 1600 by facsimile transmission. Papers should be faxed to Technology Center 1600 via the PTO Fax Center located in Crystal Mall 1. The faxing of such papers must conform to the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). The CM1 Fax Center telephone number is (703) 305-7401.

Phuong N. Huynh, Ph.D.
Patent Examiner
Technology Center 1600
February 25, 2002

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